DOI: 10.1289/EHP12775

Note to readers with disabilities: *EHP* strives to ensure that all journal content is accessible to all readers. However, some figures and Supplemental Material published in *EHP* articles may not conform to 508 standards due to the complexity of the information being presented. If you need assistance accessing journal content, please contact ehp508@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.

Supplemental Material

Global Air Pollutant Phenanthrene and Arrhythmic Outcomes in a Mouse Model

Sana Yaar, Tatiana S. Filatova, Ellie England, Shiva N. Kompella, Jules C. Hancox, David A. Bechtold, Luigi Venetucci. Denis V. Abramochkin, and Holly A. Shiels

Table of Contents

Table S1. Estimated IC₅₀, n(H) and maximal block.

Table S2. Model output of effect of Phenathrene exposure on the human ventricular action potential.

Figure S1. (**A**) A representative surface ECG from an ex vivo mouse heart showing the wave forms and intervals measured. Note the absence of a clear J-wave showing early repolarization. These were often undetectable due to interference with the QRS which is common for ex vivo mouse heart preparations and thus not quantified in this study. Lower panel shows the impact of acute phenanthrene (Phe) exposure on uncorrected QT interval (**B**), QRS duration (**C**) and P-wave duration (**D**). Average parameters calculated from isolated mouse (C57/BJ, Male, 10-weeks) hearts at baseline and after 15-minute exposure to either control solution (circle, black), 2.1 μ M Phe (square, red) or ~8 μ M Phe (triangle, dark-red) and finally following a 15-minute wash-out period. Bars are mean+/-SEM, symbols are individual hearts (N=5-7 hearts). Two-way ANOVA mixed-effects analysis with Tukey's multiple comparison test found no significant differences between groups. Numeric values are provided in Excel Table S5.

Figure S2. Monophasic action potential duration at 50% repolarization, corrected for peak-peak interval. MAP duration at 50% repolarisation, corrected for peak-peak interval (cMAPD50) at baseline and after exposure to either control solution (A) or ~8 μM phenanthrene (Phe) (B) N=6 hearts. Each point represents an individual heart, with lines showing the change after exposure. Paired Student's T-tests found no significant differences between groups (ns). Numeric values are provided in Excel Tabel S6.

Figure S3. Effect of phenanthrene on the inactivation kinetics of outward potassium currents in isolated murine ventricular myocytes. Differences in time constants of inactivation of I_{to} (A, n=6-8 cells, N=5 animals) and I_{Kur} (B, n=6-8 cells, N=6 animals) under control conditions and after exposure to different concentrations of phenanthrene (Phe). *Significant from control, ***=p<0.001, RM ANOVA. Numeric values are as follows: for I_{to} tau in ms was 16.4 ± 1.3 , 13.9 ± 1.8 , 13.7 ± 1.5 , 9.0 ± 1.1 , and 6.99 ± 0.82 ; and for I_{Kur} tau in ms was 79.8 ± 5.4 , 58.4 ± 5.9 , 42.8 ± 4.3 , 14.4 ± 2.9 , and 37.2 ± 2.9 , under control and with 1, 3, 10, and 30 μ M Phe, respectively.

References

Additional File- Excel Document